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The Role of Personality Pathology in Depression Treatment Outcome With Psychotherapy and Pharmacotherapy

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Abstract

Background—Depressed patients with comorbid personality pathology may fare worse in treatment for depression than those without this additional pathology, and comorbid personality pathology may be associated with superior response in one form of treatment relative to another, though recent findings have been mixed. We aimed to evaluate the effect of personality pathology on time to remission of patients randomly assigned to 1 of 2 treatment strategies for depression and to determine whether personality pathology moderated the effect of treatment assignment on outcome.

Method—Individuals undergoing an episode of unipolar major depression ($n = 275$) received interpersonal psychotherapy (Klerman, Weissman, Rounsaville, & Chevron, 1984) or selective serotonin reuptake inhibitor (SSRI) pharmacotherapy for depression. Depressive symptoms were measured with the HRSD-17. Remission was a mean HRSD-17 score of 7 or below over a period of 3 weeks. Personality disorders were measured according to SCID-II diagnoses, and personality pathology was measured dimensionally by summing the positive probes on the SCID-II.

Results—The presence of at least 1 personality disorder was not a significant predictor of time to remission, but a higher level of dimensionally measured personality pathology and the presence of borderline personality disorder were associated with a longer time to remission. Personality pathology did not moderate the effect of treatment assignment on time to remission.

Conclusions—The findings suggest that depressed individuals with comorbid personality pathology generally fare worse in treatment for depression, although in this report, the effect of personality pathology did not differ by the type of treatment received.

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Keywords

major depression; personality disorder; interpersonal psychotherapy; pharmacotherapy; moderators

Interpersonal psychotherapy (IPT; Klerman, Weissman, Rounsaville, & Chevron, 1984) and pharmacotherapy have proven to be generally efficacious treatments for acute depression in adult populations (e.g., Hollon et al., 2005). However, some patients are more responsive than others, prompting an interest in identifying the patient characteristics associated with treatment response. The presence of comorbid personality pathology may be one important factor. Some studies have suggested that depressed patients with comorbid personality pathology may fare worse in treatments for depression than those without these additional symptoms (Bearden, Lavelle, Buysse, Karp, & Frank, 1996; Cyranowski et al., 2004; Reich & Vasile, 1993; Shea et al., 1990), although more recent findings have been mixed (Bagby, Quilty, & Ryder, 2008; Kelly, Nur, Tyrer, & Casey, 2009). Recent reviews of this literature have differed in their conclusions, with some reports suggesting little effect of personality disorders on outcome of treatment for depression (Kool et al., 2005; Mulder, 2002) but another reporting that response to a variety of treatments for depression is poorer for those depressed patients who have comorbid personality pathology (Newton-Howes, Tyrer, & Johnson, 2006).

Despite Mulder's (2002) assertion that the most well-designed studies demonstrate little effect of personality pathology on response to depression treatment in the short term, comorbid personality pathology may be associated with maladaptive outcomes in other ways. Patients with comorbid personality pathology who fare worse in treatment tend to have earlier ages of onset and more residual symptoms of depression, as well as a more recurrent course of depression than those without personality disorders (Reich & Vasile, 1993). This may suggest a more severe clinical picture overall. The presence of a personality disorder among individuals recently remitted from depression has been shown to predict a higher likelihood of recurrence during maintenance treatment with IPT (Cyranowski et al., 2004), and Fournier et al. (2008) showed that depressed patients with personality disorders relapsed at a high rate if withdrawn from medication. This suggests that personality may be important in predicting long-term as well as acute treatment outcomes. Continued work in this area is particularly important, given the relatively high prevalence of personality pathology among individuals with major depressive disorder (Bagby et al., 2008; Hirschfeld, 1999; Pilkonis & Frank, 1988; Skodol et al., 1999) and the potential for this pathology to inhibit short-term as well as sustained recovery from depressive symptoms.

In addition to the potential prognostic implications of personality pathology on outcome across treatment modalities, it is possible that personality pathology may be associated with superior response in one form of treatment relative to another. The findings in this area are also mixed, perhaps because they have focused on a range of pharmacological and psychotherapeutic treatments. Some work has suggested that individuals with comorbid personality pathology fare more poorly with IPT than with cognitive therapy (CBT; Beck, Rush, Shaw, & Emery, 1979; Carter et al., 2011; Joyce et al., 2007), and other work found that depressed individuals with comorbid borderline personality disorder fare equally well in treatment with IPT or CBT in addition to pharmacotherapy (Bellino, Zizza, Rinaldi, & Bogetto, 2007). In comparisons of psychotherapy and pharmacotherapy, both earlier (Tyrer, Seivewright, Ferguson, Murphy, & Johnson, 1993) and more recent work (Fournier et al., 2008; Maddux et al., 2009) suggest that individuals with comorbid Axis I psychopathology and personality disorders fare better with antidepressants, and those without a personality disorder fare better with psychotherapy. Among participants in the National Institute of

Mental Health Treatment of Depression Collaborative Research Project (NIMH TDCRP), there was a trend for those without personality disorders to fare more poorly in cognitive therapy as compared to those receiving IPT, imipramine with clinical management, or placebo with clinical management (Shea et al., 1990); however, this finding did not reach conventional levels of statistical significance. Another analysis of the NIMH TDCRP showed a differential effect of psychotherapeutic treatment type (IPT vs. CT) on the reduction of depression depending on the level of specific personality dimensions (obsessive vs. avoidant; Barber & Muenz, 1996). IPT was more effective for patients with obsessive-compulsive personality disorder (OCPD), and CT was more effective for those with avoidant personality disorder (AVPD; Barber & Muenz, 1996). These findings suggest the potential importance of considering the nature of the patient's personality pathology when choosing a treatment for depression.

Some studies of depression treatment have focused on sequential treatment designs. Such designs aim to identify the specific treatment strategies that have the highest degree of success for subgroups of patients and use adjunctive treatment strategies for those who do not respond or remit with first-line treatment (e.g., Frank et al., 2000; Rush et al., 2004). It is feasible that personality pathology may impede treatment efficacy within the first weeks and months of intervention, increasing the need for adjunctive treatment if response or remission is not achieved within that time. This information may guide our understanding of which patients may be more likely to require adjunctive treatment in order to attain remission.

Some of the inconsistencies in the findings reviewed above may be a function of the numerous methods of measuring personality pathology and the variety of treatments for depression utilized in the studies examining this issue (Mulder, 2002). Still, other inconsistencies may be because much of this work focuses on the role of Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000) personality diagnoses, rather than symptoms. The reliance on cutoff criteria in the current categorical system may mask the clinical implications of subthreshold manifestations of Axis II pathology (Huprich & Bornstein, 2007), which may have a significant impact on treatment for depression and may require therapeutic intervention (Krueger, Skodol, Livesley, ShROUT, & Huang, 2007; Verheul, 2005). Moreover, categorical systems may not be well suited to addressing the heterogeneous nature of personality pathology. This is reflected in the high degree of overlap between categories and suggests that the categories may not reflect distinct diagnostic entities (Trull, Tragesser, Solhan, & Schwartz-Mette, 2007). To this end, dimensional approaches to personality pathology are being considered for the revision of *DSM-5* (Huprich & Bornstein, 2007; Krueger et al., 2007; Regier, 2007; Skodol et al., 2011; Trull et al., 2007; Verheul, 2005; Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005).

We have conducted a trial of IPT and pharmacotherapy for the treatment of patients suffering from unipolar depression, many of whom reported comorbid personality disorders and subsyndromal personality pathology. The primary findings of this trial have been reported elsewhere (Frank et al., 2008, 2011). Here, we examine whether the presence of a personality disorder diagnosis and/or the overall level of personality pathology was related to time to remission in this group of patients receiving treatment for depression with IPT (Klerman et al., 1984; Weissman, Markowitz, & Klerman, 2000, 2007) or escitalopram, a selective serotonin reuptake inhibitor (SSRI). We are interested in the potential role of personality pathology as a predictor or moderator of depression improvement. Our aims are twofold: first, to determine the role of personality disorders and level of personality pathology in determining time to remission of depression among those who remitted relatively quickly with monotherapy (i.e., in 12 or fewer weeks). Second, we aim to test whether the presence or level of personality pathology moderated the effect of treatment

assignment on time to remission within 12 weeks of treatment. Though previous work (e.g., Barber & Muenz, 1996) has examined personality pathology as a moderator of depression treatment type, to our knowledge this is the first study examining this moderation in a direct comparison of IPT and escitalopram, which may be particularly informative given the interpersonal nature of many Axis II disorders.

We hypothesized, based on these aims, that those with at least one personality disorder or those with a higher level of personality pathology measured dimensionally would have a longer time to remission from depression than those without a personality disorder and those with lower levels of personality pathology. Given the potential importance of subthreshold manifestations of personality pathology, we examined dimensional and categorical measures of personality pathology separately. In order to increase the specificity of the results, we tested the effect of individual diagnoses on time to remission if either the overall categorical or the dimensional measure of personality pathology predicted time to remission. On the basis of findings of previous work in this area comparing psychotherapy and pharmacotherapy (Fournier et al., 2008; Maddux et al., 2009; Tyrer et al., 1993), we expected that individuals with a personality disorder or a higher level of personality pathology would have shorter time to remission with antidepressant medications, and those without a personality disorder or with a lower level of personality pathology would have a shorter time to remission with IPT.

Method

Participants

The study population of 275 men and women was obtained from participants in the protocol entitled "Depression: The Search for Treatment Relevant Phenotypes" (MH65376, E. Frank, PI). This protocol was conducted at the University of Pittsburgh Medical Center's Depression and Manic Depression Prevention Program, part of the Western Psychiatric Institute and Clinic, and at the Department of Psychiatry, Neurobiology, Pharmacology, and Bio-technology at the University of Pisa, Italy. Individuals ranged in age from 18 to 65 and met criteria for a current episode of nonpsychotic unipolar major depression, as defined by the *DSM-IV-TR* (American Psychiatric Association, 2000) and a score of 15 or higher on the 17-item Hamilton Rating Scale for Depression (HRSD-17; Hamilton, 1960). Participants were excluded if they had a primary diagnosis of schizophrenia, schizoaffective disorder, bipolar I or II disorder, current anorexia or bulimia, antisocial personality disorder, or severe, uncontrolled medical illness, or if they were women who were unwilling to use an acceptable form of birth control. Individuals were also excluded if they had been unresponsive to an adequate dose of escitalopram or IPT in their current episode and if their current substance abuse or dependence (if present) was unrelated to their diagnosis of depression. Although the original study population comprised 291 individuals who received at least one treatment session, we included only the 275 participants for whom full data from the Structured Clinical Interview for *DSM-IV* Axis II (SCID-II; First, Gibbon, Spitzer, & Williams, 1997) personality disorders were available, because the remainder were administered an unreliable version of the SCID-II. The 16 individuals who were excluded were all from the Pisa site. As compared to those included in the analyses, those excluded had a lower level of education (Fisher's exact test $p = 0.003$) and a higher baseline depression score ($W = 3,016, p = .012$).

The protocol was approved by the Institutional Review Board of the University of Pittsburgh and the Ethics Committee of the Azienda Ospedaliero-Universitaria of Pisa. Study participants were informed of all study procedures and were given an opportunity to ask questions about the protocol before providing written consent to participate.

Procedures

A full description of the study procedures has been published elsewhere (Frank et al., 2008, 2011). In brief, participants who entered into the study were randomly assigned to IPT or pharmacotherapy, which consisted of the selective serotonin reuptake inhibitor (SSRI), escitalopram oxalate. Patients assigned to IPT received 45 to 50 minutes of psychotherapy per week from their therapist, and those who received SSRI met with experienced psychopharmacologists for 20 to 30 minutes per week. The study included a variable length acute treatment phase of at least 12 weeks and, once patients remitted, a 6-month continuation phase.

Participants were assessed weekly by an independent evaluator to measure depressive symptoms using the HRSD-17. Remission was defined as a mean HRSD-17 score of 7 or below over a 3-week period. If sufficient improvement had not occurred, the alternate treatment could be added at Week 6. Although participants from the original study could be given adjunctive treatment and followed for up to 64 weeks, in the current study we were interested only in the effect of personality pathology on treatment outcome during the first 12 weeks of monotherapy. This was the earliest point at which patients could move to the continuation phase and at which both SSRI and IPT monotherapy could be expected to have had a reasonable chance of success. In the current analysis, patients who did not remit with monotherapy (including those who were discontinued for clinical reasons) were censored at 12 weeks ($n = 11$), and those who dropped out prior to 12 weeks were censored at the date of dropout ($n = 32$). We assumed that those who received augmentation prior to 12 weeks would not have remitted from depression if they had continued on mono-therapy. Thus, these patients were censored at 12 weeks ($n = 91$) regardless of whether they had remission after receiving adjunctive treatment.

Therapists

IPT therapists were master's- or doctoral-level clinicians, with backgrounds in social work, psychology, psychiatry nursing, or psychiatry, who received specific training and certification in IPT for the purposes of this study. All therapists were involved in a yearlong pilot study in which they treated a minimum of two cases under individual supervision by an experienced IPT supervisor. All IPT therapists were supervised regularly during the study on the basis of audio- or videotapes of sessions. Audiotapes were rated by blind raters for adherence to IPT principles.

Measures

A diagnosis of depression was initially made with the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997) and a score of 15 or higher on the HRSD-17. Other *DSM* Axis I disorders were diagnosed in this sample with the SCID-I. Personality disorders were diagnosed with the SCID-II (First, Gibbon, et al., 1997) for *DSM-IV-TR* Axis II disorders. Individuals who met full criteria for antisocial personality disorder were excluded from the study, though individuals entering the study could have symptoms of antisocial personality disorder not meeting full diagnostic criteria. The categorical measure of personality pathology was the presence or absence of one or more personality disorders, including a diagnosis of personality disorder not otherwise specified (PDNOS). Interrater agreement at each site and between sites for the HRSD-17 was recalibrated approximately every 6 months and was maintained at an intraclass correlation coefficient (ICC) > 0.85 . With regard to the SCID-II, individuals were deemed reliable once they performed three SCID-II interviews in the presence of a seasoned clinician who evaluated their performance.

To derive a dimensional score of personality pathology for each subject, we summed the scores of individual items on the SCID-II (absent = 0, subthreshold = 1, threshold = 2). This method has been identified by others as a way to measure personality pathology dimensionally (Cyranowski et al., 2004; Feske et al., 2004; Trull et al., 2007). Given that all SCID-II probes focus on present pathology, we included only the present-focused probes of antisocial personality disorder.¹ We also considered the fact that each diagnosis comprises a varying number of probes, which means that some diagnoses could be overrepresented in the dimensional measure that contains the sum of all probes. However, given the high correlation of actual sum and weighted sum of items ($r = .998$, $p < .0001$), we used the actual sum of probes in all analyses.

Outcome Measure

We examined time to remission on monotherapy (IPT or SSRI) up to the 12-week point. This was the earliest point at which patients could move to the continuation phase and at which both treatments were expected to have had a reasonable chance of success.

Data Analysis

Analyses were performed with PASW Version 18.0 for Mac, R Foundations for Statistical Computing Version 2.12.0 for Windows 2, and SAS version 9.3. Chi-square, Wilcoxon, and t tests were conducted to compare patients with and without at least one personality disorder and those included and not included in the analyses on demographic and clinical variables. Univariate regression analyses were conducted to determine which patient variables were marginally related to the dimensional measure of personality pathology. Univariate Cox proportional hazard models were conducted to identify patient variables related to time to remission and to select covariates for use in the multivariable survival models. Multivariable Cox proportional hazard models to determine the effect of personality pathology variable on time to remission in monotherapy by 12 weeks were built by first entering the selected covariates into the model and then entering the personality pathology variable of interest. We included interaction terms in later steps of these models in order to examine whether the personality pathology variable moderated the effect of treatment assignment on time to remission. Chi-square and Fisher's exact tests examined the response rate by treatment type for each personality disorder diagnosis. Finally, we fit accelerated failure time models using the same variables included in the Cox proportional hazards model in order to obtain a more clinically relevant interpretation of the impact of personality pathology on time to depression remission.

Results

The demographic and clinical characteristics of study participants are presented in Table 1. Of the 275 individuals included in this analysis, 114 met criteria for one or more personality disorder diagnoses. Individuals assigned to SSRI treatment were more likely to have at least one personality disorder ($\chi^2 = 4.895$, $p = .027$). Individuals with and without at least one personality disorder differed on age of first depression onset category (<24, 24–50, >50 years; $\chi^2 = 11.084$, $p = .004$). Examination of the adjusted standardized residuals showed

¹For some participants ($n = 82$) who did not endorse any items pertaining to antisocial behaviors prior to the age of 15, present-focused items were typically not probed, as the participants could not have met diagnostic criteria for this disorder regardless of what these items showed. To account for this in creating the dimensional measure of personality pathology, we used three strategies: (a) we imputed scores of zero for any item that was missing for this reason, with the rationale that these participants were unlikely to endorse a present-focused probe given the fact that they did not meet criteria for early-onset conduct disorder; (b) we imputed scores of zero for any present-focused item for anyone who did not meet criteria for early-onset conduct disorder, even if his or her present-focused items were probed; (c) we excluded individuals with missing present-focused items. When we ran the hypothesis testing analyses using each strategy, the results did not show meaningful differences. Thus, because the second strategy seems to carry the least amount of bias, we chose that method to use for the remainder of the analyses.

that individuals with an age at first depression onset of less than 24 were more likely to have a personality disorder; however, individuals with an age of first depression onset of between 24 and 50 were less likely to have a personality disorder. No association was seen among individuals over 50. Table 2 shows the frequency of personality disorders in this sample by site. Individuals at the Pittsburgh site were more likely to have schizoid personality disorder (Fisher's exact $p = 0.019$) and dependent personality disorder ($\chi^2 = 3.79$, $p = 0.052$), and individuals at the Pisa site were more likely to have dependent personality disorder (Fisher's exact $p = 0.013$), although the absolute number of patients with either of these disorders was quite low.

The median dimensional personality pathology score reported by study participants was 20 (interquartile range = 21) with a mean of 22.50 ($SD = 15.24$). Because the dimensional personality pathology variable was not normally distributed, we used a square root transformation to evaluate which patient variables are related to this measure of personality pathology. Being married or living as married, $t(273) = 2.6$, $p = 0.010$; having a higher level of education (14+years), $t(273) = -2.53$, $p = 0.012$; being censored for having received adjunctive treatment, $t(273) = -2.72$, $p = 0.007$; and being at the Pittsburgh site, $t(273) = 5.06$, $p < 0.001$, were all significantly associated with higher levels of personality pathology. Age category of first depressive episode was also significantly associated with the dimensional measure of personality pathology, $F(2, 271) = 14.13$, $p < 0.0001$. Post hoc Tukey's pairwise tests showed those in the youngest age of onset category (0–23) had significantly greater levels of pathology than those age 24–50.

Effects of Personality Pathology on Time to Remission

Of the 275 participants included, 51.3% ($n = 141$) remitted by 12 weeks, and 48.7% ($n = 134$) did not remit or were lost to follow-up by this point. Median time to remission in the study as a whole was 65 days. The presence of one or more personality disorder diagnosis was not a significant predictor of time to remission either alone, $\text{Exp}(\beta) = 0.816$, 95% CI [0.581, 1.146], $p = 0.240$, or when all relevant covariates (age, gender, marital status, level of education, site, baseline depression score, the presence of an additional Axis I disorder, and age at first depressive episode) were entered into the model, $\text{Exp}(\beta) = 0.922$, 95% CI [0.649, 1.311], $p = 0.652$. However, those with a higher level of personality pathology on the dimensional measure had a significantly longer time to remission, $\text{Exp}(\beta) = 0.974$, 95% CI [0.962, 0.987], $p < 0.001$, than those with a lower level of pathology, even after all relevant covariates were added to the model, $\text{Exp}(\beta) = 0.984$, 95% CI [0.971, 0.997], $p = 0.018$. The multivariate model including the dimensional measure is shown in Table 3.

It is important to note that although the antisocial portion of the SCID-II was completed at screen in order to exclude those who met criteria for this diagnosis, two participants endorsed full criteria for antisocial personality disorder when the assessment was repeated at visit 2, the time when the entire SCID-II was completed. The results did not change meaningfully when we repeated the analyses without these two individuals.

Given the possibility that personality pathology symptom count may be a proxy for general severity or level of functioning, we sought to test whether the significant association of the dimensional measure of personality pathology still held when controlling for such a measure. We did not include the Global Assessment of Functioning or Global Assessment Scale in this study, so our best proxies for general level of severity are the 17- and 25-item HRSD (Thase, Carpenter, Kupfer, & Frank, 1991). Both the 17-item baseline HRSD score and the dimensional level of personality were significant predictors of time to remission in the original model (see Table 3). The HRSD-25 was a strong predictor of time to remission in a univariate Cox model, $\text{Exp}(\beta) = 0.941$, 95% CI [0.908, 0.976], $p = .001$, but higher levels of personality pathology were still significantly associated with a longer time to

remission from depression even when this measure of severity was added to the model, $\text{Exp}(\beta) = 0.985$, 95% CI [0.972, 0.999], $p = .031$.

Because the dimensional measure of personality pathology significantly predicted time to remission, we conducted Cox proportional hazard analyses for the dimensional measure of each diagnosis in order to determine which personality disorder(s) was driving this effect. Only the dimensional level of borderline personality disorder significantly predicted time to remission, $\text{Exp}(\beta) = 0.939$, 95% CI [0.888, 0.993], $p = .027$, with a greater level of borderline pathology associated with a longer time to remission. All other dimensional personality variables were unrelated to time to remission when separated by diagnosis ($ps > .05$).

Moderating Effects of Personality Pathology

Our second main hypothesis concerned whether individuals with a personality disorder or a greater level of personality pathology would have a shorter time to remission with antidepressant medications, and those without a personality disorder or a lower level of personality pathology would have a shorter time to remission with IPT. The treatment-by-personality interaction term was not significant in the Cox regression analysis of time to remission using either the categorical, $\text{Exp}(\beta) = 1.043$, 95% CI [0.515, 2.113], $p = .906$, or the dimensional, $\text{Exp}(\beta) = 0.995$, 95% CI [0.971, 1.02], $p = .710$, measures of personality pathology.² Because power to detect a moderating effect in a Cox regression model may be limited with a sample size of 275, we examined the response rates of individuals with each type of personality disorder by treatment type using chi-square and Fisher's exact tests (see Table 4). Significant differences in response rates by treatment type were not found, supporting the results of our Cox regression analyses.³

Estimation of Relative Survival Time Based on Dimensional Personality Pathology Score

Given that the dimensional measure of personality pathology significantly predicted time to remission from depression, we sought to increase the clinical utility of this measure by estimating the relative increase in remission time associated with increasing levels of pathology. To do so, we conducted parametric accelerated failure-time models using the SAS LIFEREG procedure. This method has been used previously (e.g., Ilardi, Craighead, & Evans, 1997) to estimate the effect of Axis II pathology on time to depressive relapse. On the basis of prior research in this area (Ilardi et al., 1997), we initially hypothesized a Weibull baseline survival function; the resulting scale parameter differed significantly from 1 (scale = 0.542), 95% CI [0.473, 0.623], confirming the use of this distribution in our sample.

When all relevant covariates were entered into the model, the dimensional measure of personality pathology was significantly associated with time to remission, $\text{Exp}(\beta) = 1.01$, $p = .005$, indicating that the median time to depression remission for a given individual is expected to be 1.01 times greater than that of a similar individual with a one-unit-lower dimensional measure of personality pathology (a 1% increase). Stated differently, the median time to depression remission for a similar individual is expected to be 1.11 times

²We attempted to reevaluate our hypotheses using a measure of personality pathology that was designed to be dimensional, the Schedule for Non-Adaptive and Adaptive Personality (SNAP). This data was collected only for patients at Pittsburgh, rather than among the entire sample, because the SNAP has not been translated into Italian. We retested our hypotheses using the SNAP without obtaining significant results.

³Given the interpersonal nature of IPT, we compared interpersonal-versus affect-related symptoms by testing the hypotheses that those with obsessive-compulsive PD (OCPD) would be better treated with IPT than would those with avoidant PD (AVPD) and that PD type (avoidant or obsessive-compulsive) would moderate the effect of treatment assignment on time to remission (similar to that conducted by Barber & Muenz, 1996, using data from the TDCRP study). As Barber and Muenz did, we excluded those with AVPD and OCPD in the analyses. Neither of the hypotheses was supported by our data.

that of an individual with a 10-unit-lower dimensional measure of personality pathology (a 11% increase). Other significant covariates included site, $\text{Exp}(\beta) = 0.691$, $p = .005$; baseline HRSD-17 score, $\text{Exp}(\beta) = 1.039$, $p = .005$; and marital status, $\text{Exp}(\beta) = 0.742$, $p = .003$. In particular, the median time to depression remission of an individual from Pisa is expected to be 0.691 times that of a similar individual from Pittsburgh (a 30.9% decrease); the median time to depression remission of a given individual is expected to be 1.039 times that of a similar individual with a one-unit-lower baseline HRSD-17 score (a 3.9% increase); and the median time to depression remission of an individual who is either married or living as married is expected to be 0.742 times that of a similar individual who is neither married nor living as married (a 25.8% decrease). Full model results are given in Table 5.

Discussion

Our primary aim in this paper was to examine the effect of personality disorders and continuously measured personality pathology on time to remission within 12 weeks in a sample of adults receiving treatment for depression and to determine whether personality pathology was associated with differential response to IPT and escitalopram. In this study we found that a higher level of continuously measured personality pathology, but not categorical personality diagnosis, was related to a longer time to remission from depression. Because we evaluated the effect of personality pathology on relatively short-term monotherapy, the results suggest that individuals with higher levels of pathology may have an increased need for adjunctive treatment strategies. In contrast to some prior studies, personality pathology did not moderate the effect of treatment assignment on time to remission in the present trial. The predictive power of dimensional personality pathology was not accounted for by any of the variables that were associated with likelihood of remission in their own right. Moreover, the main effect of personality pathology was significant above and beyond the impact of HRSD-25 depression severity at baseline, suggesting that personality pathology symptom count is not simply a proxy for general level of severity.

With regard to the type of personality pathology that may be most clinically relevant, we found that high levels of borderline personality pathology may be the most relevant to how well a patient will fare in short-term treatment. Thus, personality pathology and in particular borderline pathology may be at least partially responsible for impeding the response of some patients to the first intervention for depression. If so, for some patients it may be useful to incorporate strategies aimed at addressing interpersonal interactions that are more specific to comorbid borderline personality pathology and depression, such as those used in IPT for borderline personality disorder (e.g., Bellino, Rinaldi, & Bogetto, 2010; Markowitz, Skodol, & Bleiberg, 2006).

The lack of a moderation effect is consistent with the primary report of this trial, which indicated few moderators of IPT versus pharmacotherapy outcome (Frank et al., 2011). Yet, some previous reports have indicated that individuals with personality pathology have better outcomes with medication than with some forms of psychotherapy (Fournier et al., 2008; Maddux et al., 2009; Tyrer et al., 1993). There may be several reasons for this discrepancy, including the inclusion criteria for the previous studies and the form of psychotherapy being studied. For example, Tyrer et al. studied individuals with generalized anxiety disorder, panic disorder, and dysthymic disorder, whereas the present study focused on moderately depressed adults. In their study of the effects of personality pathology on treatment outcome of depression, Fournier et al. excluded individuals with antisocial, schizotypal, or borderline PD, but our sample excluded only antisocial PD. Second, to our knowledge this is the first comparison of IPT and escitalopram. Earlier studies examined cognitive therapy and self-help. IPT focuses on improving interpersonal functioning as a way of ameliorating

depression, a focus that may be particularly applicable to those with Axis II pathology. Indeed, past studies have shown that depressed individuals who receive maintenance treatment with IPT may experience continuous improvement in personality pathology over 2 years of treatment (Cyranowski et al., 2004). This focus on interpersonal dysfunction may explain why IPT performed as well as medication in this trial for individuals with higher levels of personality pathology. Last, the effect of therapy up to 12 weeks may have been too short of a time in which to see significant differences between the treatments; however, this was the longest reasonable period to examine the two initial monotherapies uncontaminated by augmentation with the other treatment.

The present report also provides another example of the differences in findings using categorical and dimensional personality disorder symptom scores. Although the significant findings may be the result of the increased power that comes with a dimensional measure, it may also be the case that symptoms of personality pathology that do not meet current diagnostic criteria may still be of sufficient importance to affect treatment for depression. This could be especially true when we consider that individuals may endorse personality pathology across multiple disorders, which, when taken together, may reveal personality dysfunction that affects treatment of depression outcome but is not captured by categorical diagnoses. The individual PDNOS diagnosis is designed to capture such symptoms. However, it was virtually never used by the study clinicians, despite their awareness of its availability. With only two participants diagnosed with PDNOS in this sample, we could not consider that category to be a valid one, and this limited our ability to examine the effects of this diagnosis. Though it may be possible to assign a diagnosis of PDNOS based on counts of individual criteria, doing so in the present analyses would more closely approximate the dimensional measure of personality pathology that we created by summing the individual probes, rather than assigning a true diagnosis of PDNOS.

These findings also provide empirical support for the notion that a dimensional measure of personality pathology may be useful in clinical decision making and treatment planning (Verheul, 2005). Here, every additional probe endorsed on the SCID-II was associated with a 2% decreased likelihood of remitting from depression on monotherapy. In addition, we estimated the increased median time to remission associated with every 10-unit increase in this measure. A 10-point increase in this measure, approximately one half the interquartile range, was associated with an 11% longer expected median time to depression remission. This finding may be helpful to clinicians treating individuals with comorbid personality pathology who not only want to know the likelihood of remitting from depression but also the relative length of treatment required for remission based on the level of pathology that is present. The utility of a dimensional measure may also be related to some interesting site differences we observed. Though individuals at the two sites did not differ on the presence of at least one personality disorder, those at Pittsburgh had a higher overall level of personality pathology based on the sum of the individual SCID-II probes. Previous reports (Frank et al., 2011) of this sample noted that patients at the Pittsburgh site were more likely to have a longer history of depression with more recurrent episodes, and in the current analyses those at the Pisa site were more likely to have remitted by 12 weeks ($\chi^2 = 29.713$, $p < .0001$), all of which may indicate a greater severity of illness in the Pittsburgh sample. Our results suggest that depression severity may be a proxy for level of personality pathology and that personality pathology may be a better indicator of depression severity than the presence of an Axis II diagnosis.

These results are also important for the question of the clinical utility of the revision of the personality disorders section of the *DSM-5* that is ongoing. Though the proposed reconceptualization has not been finalized, the results of the current analyses suggest that borderline personality pathology is a particularly important contributor to the outcome of

treatment of depression, and valuable information regarding the outcome of this study may be lost by excluding this diagnosis from future iterations of the *DSM*. This may be due to the affective and interpersonal instability experienced by individuals with this personality disorder. On the other hand, future replications of null findings for some personality disorders (e.g., avoidant and obsessive-compulsive) across treatment modalities would begin to call into question the utility of these disorders for those interested in predicting depression treatment response. Individuals interested in predicting time to remission from depression might not need to measure these parameters, even though these two diagnoses were even more prevalent than borderline PD in our sample. Most of all, the predictive ability of the dimensional personality pathology variable in the current analyses supports the dimensional aspect of the proposed *DSM-5* hybrid model (Skodol et al., 2011).

Limitations

Although only four of the 552 patients screened for this trial were excluded because of antisocial PD (ASPD) based on concerns about being able to appropriately manage such patients within the context of the study protocol, their absence is an important limitation of this study. This practice is not uncommon in clinical trials; however, the exclusion of such participants may be more relevant to this report because of our focus on the impact of personality disorders on treatment outcome. Thus, we cannot draw conclusions about the impact of full criterion ASPD. However, those with symptoms of ASPD who did not meet diagnostic criteria for this disorder as well as two individuals who were later found to have ASPD were included in the study. Thus, the dimensional measure of personality pathology provides some indication of the impact of antisocial personality pathology on depression treatment outcome. Although IPT performed as well as pharmacotherapy for individuals with a personality disorder in this study, we cannot know whether both treatments performed equally well or equally poorly for these patients because we do not have a placebo comparison group.

Another limitation is the less-than-ideal psychometric properties of the dimensional measure of personality pathology used here. Though this method has been used elsewhere (Cyranowski et al., 2004; Feske et al., 2004; Trull et al., 2007), this dimensional method includes multiple probes that may measure similar personality traits, providing a more severe picture of personality pathology than may be truly present. Future investigations might benefit from choosing to utilize a dimensional measure of the SCID-II that aims to reduce the redundancy in the individual probes.

A third limitation grows from that fact that any dimensional measure is likely to have more power to detect an effect than does a categorical one, which may have contributed to the different findings with the two measures of personality pathology. Thus, we cannot be certain whether the greater impact of personality pathology on time to remission with the dimensional measure is based on the true impact of pathology considered in this way or on the increased power to detect this effect. It is also important to note that age of onset of depression was related to the presence of personality pathology in our sample. This suggests that personality pathology may be a proxy for depression chronicity, which may be related to likelihood of remission on its own. Still, level of personality pathology significantly predicted time to remission from depression in our sample even when controlling for age of depression onset and baseline depression, and we did not find age of onset to be related to time to remission in the main outcome paper of this study using a slightly larger sample. Finally, the generalizability of these findings is limited because the study population was primarily made up of Caucasian individuals and because we considered the effects of personality pathology on treatment only up to 12 weeks.

Conclusion

We examined the effect of personality pathology on relatively short-term treatment outcome of depression in a sample of patients receiving IPT or pharmacotherapy for depression, some of whom also reported comorbid personality pathology. The findings support our hypothesis that a higher level of personality pathology is related to longer time to remission from depression, with borderline personality pathology carrying the majority of this effect. Individuals with personality pathology had similar outcomes with IPT and escitalopram, in contrast to our hypothesis that those with personality pathology would fare better in treatment with pharmacotherapy than psychotherapy. This may, in part, reflect the interpersonal focus of IPT, which may be particularly applicable to individuals with Axis II pathology.

The findings contribute to our understanding of the effects of personality pathology on treatment of depression when comorbid personality pathology is present. They highlight the potential need for adjunctive treatment strategies among those with greater levels of personality pathology, and they underscore the importance of symptoms of personality pathology that are insufficient to meet diagnostic criteria. Clinicians treating individuals with depression may want to pay particular attention to a patient's level of borderline personality pathology in predicting how well he or she may fare in treatment. Moreover, our findings suggest that clinicians may be able to gauge relative time to remission from depression based on the level of personality pathology the patient endorses, using the findings reported here.

Future work should continue to focus on which patient characteristics best predict outcome of treatment for depression and how these may vary based on the specific nature of the patient's personality pathology and the nature of the treatment being offered. Future studies should also focus on the proposed dimensional models of personality pathology to identify those that are the most reliable and the most useful in clinical decision making. Last, future work should bear in mind the role of personality pathology in the outcome of treatment of depression as the revisions of *DSM-5* are finalized. Our findings suggest that important information might be lost if those disorders that significantly predict the likelihood of depression are excluded from the next iteration of the *DSM*. Investigators should continue to evaluate the role of personality pathology as currently conceptualized and its relationship to constructs such as overall severity and chronicity of depression as the changes are finalized. All of this work would contribute to our understanding of the most important patient characteristics that predict various aspects of response to a variety of treatments for depression.

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References

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed., text rev.. Author; Washington, DC: 2000.
- Bagby RM, Quilty LC, Ryder AC. Personality and depression. *Canadian Journal of Psychiatry/Revue canadienne de psychiatrie*. 2008; 53:14–25.
- Barber JP, Muenz LR. The role of avoidance and obsessiveness in matching patients to cognitive and interpersonal psychotherapy: Empirical findings from the Treatment for Depression Collaborative

- Research Program. *Journal of Consulting and Clinical Psychology*. 1996; 64:951–958. doi: 10.1037/0022-006X.64.5.951. [PubMed: 8916624]
- Bearden C, Lavelle N, Buysse D, Karp J, Frank E. Personality pathology and time to remission in depressed outpatients treated with interpersonal psychotherapy. *Journal of Personality Disorders*. 1996; 10:164–173. doi:10.1521/pedi.1996.10.2.164.
- Beck, AT.; Rush, AJ.; Shaw, BF.; Emery, G. *Cognitive therapy of depression*. Guilford Press; New York, NY: 1979.
- Bellino S, Rinaldi C, Bogetto F. Adaptation of interpersonal psychotherapy to borderline personality disorder: A comparison of combined therapy and single pharmacotherapy. *Canadian Journal of Psychiatry/Revue canadienne de psychiatrie*. 2010; 55:74–81.
- Bellino S, Zizza M, Rinaldi C, Bogetto R. Combined therapy of major depression with concomitant borderline personality disorder: Comparison of interpersonal and cognitive psychotherapy. *Canadian Journal of Psychiatry/Revue canadienne de psychiatrie*. 2007; 52:718–725.
- Carter JD, Luty SE, McKenzie JM, Mulder RT, Frampton CM, Joyce PR. Patient predictors of response to cognitive behaviour therapy and interpersonal psychotherapy in a randomized clinical trial for depression. *Journal of Affective Disorders*. 2011; 128:252–261. doi:10.1016/j.jad.2010.07.002. [PubMed: 20674982]
- Cyranowski JM, Frank E, Winter E, Rucci P, Novick D, Pilkonis PA, Kupfer J. Personality pathology and outcome in recurrently depressed women over 2 years of maintenance interpersonal psychotherapy. *Psychological Medicine*. 2004; 34:659–669. doi:10.1017/S0033291703001661. [PubMed: 15099420]
- Feske U, Mulsant BH, Pilkonis PA, Soloff P, Dolata D, Sackeim HA, Haskett HA. Clinical outcome of ECT in patients with major depression and comorbid borderline personality disorder. *American Journal of Psychiatry*. 2004; 161:2073–2080. doi:10.1176/appi.ajp.161.11.2073. [PubMed: 15514409]
- First, MB.; Gibbon, M.; Spitzer, RL.; Williams, JBW. *Structured Clinical Interview for DSM–IV Axis II Personality Disorders*. American Psychiatric Publishing; Washington, DC: 1997.
- First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. *Structured Clinical Interview for DSM–IV Axis I Disorders (SCID-I)*. American Psychiatric Publishing; Washington, DC: 1997.
- Fournier JC, DeRubeis RJ, Shelton RC, Gallop R, Amsterdam JD, Hollon SD. Antidepressant medications v. cognitive therapy in people with depression with or without personality disorder. *British Journal of Psychiatry*. 2008; 192:124–129. doi:10.1192/bjp.bp.107.037234. [PubMed: 18245030]
- Frank E, Cassano GB, Rucci P, Fagiolini A, Maggi L, Kraemer HC, Forgiione RN. Addressing the challenges of a cross-national investigation: Lessons from the Pittsburgh–Pisa study of treatment-relevant phenotypes of unipolar depression. *Clinical Trials*. 2008; 5:253–261. doi: 10.1177/1740774508091965. [PubMed: 18559415]
- Frank E, Cassano GB, Rucci P, Thompson WK, Kraemer HC, Fagiolini A, Forgiione RN. Predictors and moderators of time to remission of major depression with interpersonal psychotherapy and SSRI pharmacotherapy. *Psychological Medicine*. 2011; 41:151–162. doi: 10.1017/S0033291710000553. [PubMed: 20380782]
- Frank E, Grochocinski VJ, Spanier CA, Buysse DJ, Cherry CR, Houck PR, Kupfer DJ. Interpersonal psychotherapy and antidepressant medication: Evaluation of a sequential treatment strategy in women with recurrent major depression. *Journal of Clinical Psychiatry*. 2000; 61:51–57. doi: 10.4088/JCP.v61n0112. [PubMed: 10695647]
- Hamilton M. A rating scale for depression. *Journal of Neurology, Neurosurgery & Psychiatry*. 1960; 23:56–62. doi:10.1136/jnnp.23.1.56.
- Hirschfeld RMA. Personality disorders and depression: Comorbidity. *Depression and Anxiety*. 1999; 10:142–146. doi:10.1002/(SICI)1520-6394(1999)10:4<142::AID-DA2>3.0.CO;2-Q. [PubMed: 10690575]
- Hollon SD, Jarrett DB, Nierenberg AA, Thase ME, Madhukar T, Rush J. Psychotherapy and medication in the treatment of adult and geriatric depression: Which monotherapy or combined treatment? *Journal of Clinical Psychiatry*. 2005; 66:455–468. doi:10.4088/JCP.v66n0408. [PubMed: 15816788]

- Huprich SK, Bornstein RF. An overview of issues related to categorical and dimensional models of personality disorder assessment. *Journal of Personality Assessment*. 2007; 89:3–15. doi: 10.1080/00223890701356904. [PubMed: 17604530]
- Ilardi SS, Craighead WE, Evans DD. Modeling relapse in unipolar depression: The effects of dysfunctional cognitions and personality disorder. *Journal of Consulting and Clinical Psychology*. 1997; 65:381–391. doi:10.1037/0022-006X.65.3.381. [PubMed: 9170761]
- Joyce PR, McKenzie JM, Carter JD, Rae AM, Luty SE, Frampton CM, Mulder RT. Temperament, character and personality disorders as predictors of response to interpersonal psychotherapy and cognitive-behavioural therapy for depression. *British Journal of Psychiatry*. 2007; 190:503–508. doi:10.1192/bjp.bp.106.024737. [PubMed: 17541110]
- Kelly BD, Nur UA, Tyrer P, Casey P. Impact of severity of personality disorder on the outcome of depression. *European Psychiatry*. 2009; 24:322–326. doi:10.1016/j.eurpsy.2008.12.004. [PubMed: 19195850]
- Klerman, GL.; Weissman, MM.; Rounsaville, BJ.; Chevron, ES. Interpersonal psychotherapy of depression. Basic Books; New York: NY: 1984.
- Kool S, Schoevers R, de Maat S, Van R, Molenaar P, Vink A, Dekker J. Efficacy of pharmacotherapy in depressed patients with and without personality disorders: A systematic review and meta-analysis. *Journal of Affective Disorders*. 2005; 88:269–278. doi:10.1016/j.jad.2005.05.017. [PubMed: 16165217]
- Krueger RF, Skodol AE, Livesley WJ, Shrout PE, Huang Y. Synthesizing dimensional and categorical approaches to personality disorders: Refining the research agenda for *DSM-V* Axis II. *International Journal of Methods in Psychiatric Research*. 2007; 16(Suppl. 1):S65–S73. doi: 10.1002/mpr.212. [PubMed: 17623397]
- Maddux RE, Riso LP, Klein DN, Markowitz JC, Rothbaum BO, Arnow BA, Thase ME. Select comorbid personality disorders and the treatment of chronic depression with nefazodone, targeted psychotherapy, or their combination. *Journal of Affective Disorders*. 2009; 117:174–179. doi: 10.1016/j.jad.2009.01.010. [PubMed: 19217168]
- Markowitz JC, Skodol AE, Bleiberg K. Interpersonal psychotherapy for borderline personality disorder: Possible mechanisms of change. *Journal of Clinical Psychology*. 2006; 62:431–444. doi: 10.1002/jclp.20242. [PubMed: 16470711]
- Mulder RT. Personality pathology and treatment outcome in major depression: A review. *American Journal of Psychiatry*. 2002; 159:359–371. doi:10.1176/appi.ajp.159.3.359. [PubMed: 11869996]
- Newton-Howes G, Tyrer P, Johnson T. Personality disorder and the outcome of depression: Meta-analysis of published studies. *British Journal of Psychiatry*. 2006; 188:13–20. doi:10.1192/bjp.188.1.13. [PubMed: 16388064]
- Pilkonis PA, Frank E. Personality pathology in recurrent depression: Nature, prevalence, and relationship to treatment response. *American Journal of Psychiatry*. 1988; 145:435–441. [PubMed: 3348446]
- Regier DA. Dimensional approaches to psychiatric classification: Refining the research agenda for *DSM-V*: An introduction. *International Journal of Methods in Psychiatric Research*. 2007; 16(Suppl. 1):S1–S5. doi:10.1002/mpr.209. [PubMed: 17623390]
- Reich JH, Vasile RG. Effect of personality disorders on the treatment outcome of Axis I conditions: An update. *Journal of Nervous and Mental Disease*. 1993; 181:475–484. doi: 10.1097/00005053-199308000-00002. [PubMed: 8103074]
- Rush AJ, Fava M, Wisniewski SR, Lavori PW, Trivedi MH, Sackeim HA, Niederehe G. Sequenced treatment alternatives to relieve depression (STAR*D): Rationale and design. *Controlled Clinical Trials*. 2004; 25:119–142. doi:10.1016/S0197-2456(03)00112-0. [PubMed: 15061154]
- Shea MT, Pilkonis PA, Beckham E, Collins JF, Elkin I, Sotsky SM, Docherty JP. Personality disorders and treatment outcome in the NIMH Treatment of Depression Collaborative Research Program. *American Journal of Psychiatry*. 1990; 147:711–718. [PubMed: 2343912]
- Skodol AE, Bender DS, Morey LC, Clark LA, Oldham JM, Alarcon RD, Siever LJ. Personality disorder types proposed for *DSM-5*. *Journal of Personality Disorders*. 2011; 25:136–169. doi: 10.1521/pedi.2011.25.2.136. [PubMed: 21466247]

- Skodol AE, Stout RL, McGlashan TH, Grilo CM, Gunderson JG, Shea MT, Oldham JM. Co-occurrence of mood and personality disorders: A report from the Collaborative Longitudinal Personality Disorders Study (CLPS). *Depression and Anxiety*. 1999; 10:175–182. doi:10.1002/(SICI)1520-6394(1999)10:4<175::AID-DA6>3.0.CO;2-2. [PubMed: 10690579]
- Thase ME, Carpenter L, Kupfer DJ, Frank E. Clinical significance of reversed vegetative subtypes of recurrent major depression. *Psychopharmacology Bulletin*. 1991; 27:17–22. [PubMed: 1862201]
- Trull TJ, Tragesser SL, Solhan M, Schwartz-Mette R. Dimensional models of personality disorder: Diagnostic and statistical manual of mental disorders fifth edition and beyond. *Current Opinion in Psychiatry*. 2007; 20:52–56. doi:10.1097/YCO.0b013e328010c838. [PubMed: 17143083]
- Tyrer P, Seivewright N, Ferguson B, Murphy S, Johnson AL. The Nottingham study of neurotic disorder. Effect of personality status on response to drug treatment, cognitive therapy and self-help over two years. *British Journal of Psychiatry*. 1993; 162:219–226. doi: 10.1192/bjp.162.2.219. [PubMed: 8435693]
- Verheul R. Clinical utility of dimensional models for personality pathology. *Journal of Personality Disorders*. 2005; 19:283–302. doi:10.1521/pedi.2005.19.3.283. [PubMed: 16175737]
- Weissman, MM.; Markowitz, JC.; Klerman, GL. Comprehensive guide to interpersonal psychotherapy. Basic Books; New York, NY: 2000.
- Weissman, MM.; Markowitz, JC.; Klerman, GL. Clinician's quick guide to interpersonal psychotherapy. Oxford University Press; New York, NY: 2007.
- Widiger TA, Simonsen E, Krueger R, Livesley J, Verheul R. Personality disorder research agenda for the *DSM-V*. *Journal of Personality Disorders*. 2005; 19:315–338. doi:10.1521/pedi.2005.19.3.315. [PubMed: 16175740]

Table 1

Characteristics of the Sample by Presence of a Personality Disorder

Characteristic	Personality disorder(s) present (41.5%, <i>n</i> = 114)	Personality disorder(s) absent (58.5%, <i>n</i> = 161)	Total	<i>t</i> , χ^2 , or <i>W</i>
Mean age (<i>SD</i>)	39.11 (11.93)	39.68 (12.24)	39.44 (12.10)	0.38
Sex (female)	66.7%	75.2%	71.6%	2.42
Ethnicity (Caucasian)	86.0%	87.0%	86.5%	0.06
Education (14+ years)	50.9%	51.6%	51.3%	0.01
Married or living with a partner	34.2%	42.9%	39.3%	2.09
Age at first episode ^a				11.08 ^{**}
0–23 ^b	57.5%	38.5%	46.4%	
24–50 ^c	39.8%	53.4%	47.8%	
51 +	2.7%	8.1%	5.8%	
Previous depressive episodes				9.07
1	22.8%	36.0%	30.5%	
2	27.2%	17.4%	21.5%	
3	14.9%	16.1%	15.6%	
4	9.6%	12.4%	11.3%	
5 +	25.4%	18.0%	21.1%	
Censored for adjunctive treatment added	42.9%	40.8%	41.5%	0.74
Median baseline HRSD-17 (1st–3rd quartiles)	19.0 (17.0–22.0)	19.0 (17.0–22.5)	19.0 (17.0–22.0)	9,600.00
Other Axis I disorder(s) present	48.2%	39.8%	43.3%	1.96
Site				0.78
Pittsburgh	58.8%	53.4%	55.6%	
Pisa	41.2%	46.6%	44.4%	
SSRI treatment	57.0%	43.5%	49.1%	4.90 [*]
Remitted at 12 weeks	47.4%	54.0%	51.3%	1.19

Note. HRSD-17 = Hamilton Rating Scale for Depression; SSRI = selective serotonin reuptake inhibitor; PD = personality disorder.

^a*n* = 214.

^bPD present > PD absent.

^cPD present < PD absent.

^{*}*p* < .05.

^{**}*p* < .01.

Table 2

Distribution of Personality Disorder Diagnosis by Site (%)

Personality disorder	Pittsburgh site (n = 153)	Pisa site (n = 122)	Total (n = 275)	Statistic
Avoidant	22.2	13.1	18.2	$\chi^2 = 3.79^*$
Dependent	1.3	7.4	4.0	Fisher's exact $p = 0.013$
Obsessive compulsive	19.0	19.7	19.3	$\chi^2 = 0.02$
Paranoid	8.5	3.3	6.2	Fisher's exact $p = 0.083$
Schizotypal	0.0	0.0	0.0	N/A
Schizoid	4.6	0.0	2.5	Fisher's exact $p = 0.019$
Narcissistic	1.3	0.0	0.7	Fisher's exact $p = 0.505$
Borderline	5.2	7.5	6.2	$\chi^2 = 0.54$
Histrionic	0.0	1.6	0.7	Fisher's exact $p = 0.196$
Antisocial	1.3	0.0	0.7	Fisher's exact $p = 0.505$
PDNOS	1.3	0.0	0.7	Fisher's exact $p = 0.505$

Note. PDNOS = personality disorder not otherwise specified; N/A = not available.

* $p < .10$.

Table 3

Relationship Between Dimensional Measure of Personality Pathology and Treatment Outcome

Variable	Exp(β) [95% CI]	<i>p</i>
Age	0.993 [0.98, 1.01]	<i>ns</i>
Sex	1.02 [0.68, 1.53]	<i>ns</i>
Married	1.56 [1.08, 2.25]	.018
Education	0.90 [0.58, 1.38]	<i>ns</i>
Age of depression onset (0–23 as reference group)		<i>ns</i>
24–50	1.24 [0.84, 1.82]	<i>ns</i>
51 +	1.11 [0.50, 2.47]	<i>ns</i>
Baseline 17-item HRSD score	0.94 [0.90, 0.99]	.019
Other Axis I disorder	0.81 [0.57, 1.17]	<i>ns</i>
Site	1.82 [1.13, 2.91]	.013
Dimensional personality pathology	0.98 [0.97, 0.997]	.018

Note. HRSD-17 = Hamilton Rating Scale for Depression; CI = confidence interval; *ns* = nonsignificant.

Table 4**Twelve-Week Remission Rate by Treatment Condition**

Personality disorder	Treatment	Remitted	Not remitted	Statistic
Avoidant (<i>n</i> = 50)	IPT	52.6% (<i>n</i> = 10)	47.4% (<i>n</i> = 9)	$\chi^2 = 0.26$
	SSRI	45.2% (<i>n</i> = 14)	54.8% (<i>n</i> = 17)	
	Total	48.0% (<i>n</i> = 24)	52.0% (<i>n</i> = 26)	
Dependent (<i>n</i> = 11)	IPT	50.0% (<i>n</i> = 2)	50.0% (<i>n</i> = 2)	Fisher's exact <i>p</i> = 1.00
	SSRI	42.9% (<i>n</i> = 3)	57.1% (<i>n</i> = 4)	
	Total	45.5% (<i>n</i> = 5)	54.5% (<i>n</i> = 6)	
Obsessive-compulsive (<i>n</i> = 53)	IPT	41.7% (<i>n</i> = 10)	58.3% (<i>n</i> = 14)	$\chi^2 = 2.19$
	SSRI	62.1% (<i>n</i> = 18)	37.9% (<i>n</i> = 11)	
	Total	52.8% (<i>n</i> = 28)	47.2% (<i>n</i> = 25)	
Paranoid (<i>n</i> = 17)	IPT	42.9% (<i>n</i> = 3)	57.1% (<i>n</i> = 4)	Fisher's exact <i>p</i> = .647
	SSRI	60.0% (<i>n</i> = 6)	40.0% (<i>n</i> = 4)	
	Total	52.9% (<i>n</i> = 9)	47.1% (<i>n</i> = 8)	
Schizotypal (<i>n</i> = 0)	IPT	0.0% (<i>n</i> = 0)	0.0% (<i>n</i> = 0)	N/A
	SSRI	0.0% (<i>n</i> = 0)	0.0% (<i>n</i> = 0)	
	Total	0.0% (<i>n</i> = 0)	0.0% (<i>n</i> = 0)	
Schizoid (<i>n</i> = 7)	IPT	0.0% (<i>n</i> = 0)	100.0% (<i>n</i> = 4)	N/A
	SSRI	0.0% (<i>n</i> = 0)	100.0% (<i>n</i> = 3)	
	Total	0.0% (<i>n</i> = 0)	100.0% (<i>n</i> = 7)	
Narcissistic (<i>n</i> = 2)	IPT	50.0% (<i>n</i> = 1)	50.0% (<i>n</i> = 1)	N/A
	SSRI	0.0% (<i>n</i> = 0)	0.0% (<i>n</i> = 0)	
	Total	50.0% (<i>n</i> = 1)	50.0% (<i>n</i> = 1)	
Borderline (<i>n</i> = 17)	IPT	33.3% (<i>n</i> = 3)	66.7% (<i>n</i> = 6)	Fisher's exact <i>p</i> = .347
	SSRI	62.5% (<i>n</i> = 5)	37.5% (<i>n</i> = 3)	
	Total	47.1% (<i>n</i> = 8)	52.9% (<i>n</i> = 9)	
Histrionic (<i>n</i> = 2)	IPT	100.0% (<i>n</i> = 1)	0.0% (<i>n</i> = 0)	Fisher's exact <i>p</i> = 1.00
	SSRI	0.0% (<i>n</i> = 0)	100% (<i>n</i> = 1)	
	Total	50.0% (<i>n</i> = 1)	50.0% (<i>n</i> = 1)	
Antisocial (<i>n</i> = 2)	IPT	50.0% (<i>n</i> = 1)	50.0% (<i>n</i> = 1)	N/A
	SSRI	0.0% (<i>n</i> = 0)	0.0% (<i>n</i> = 0)	
	Total	50.0% (<i>n</i> = 1)	50.0% (<i>n</i> = 1)	
PDNOS (<i>n</i> = 2)	IPT	0.0% (<i>n</i> = 0)	0.0% (<i>n</i> = 0)	N/A
	SSRI	0.0% (<i>n</i> = 0)	100.0% (<i>n</i> = 2)	
	Total	0.0% (<i>n</i> = 0)	100.0% (<i>n</i> = 2)	
None (<i>n</i> = 161)	IPT	53.8% (<i>n</i> = 49)	46.2% (<i>n</i> = 42)	$\chi^2 = 0.003$
	SSRI	54.3% (<i>n</i> = 38)	45.7% (<i>n</i> = 32)	
	Total	54.0% (<i>n</i> = 87)	46.0% (<i>n</i> = 74)	

Note. IPT = interpersonal psychotherapy; SSRI = selective serotonin reuptake inhibitor; PDNOS = personality disorder not otherwise specified; N/A = not available.

Table 5

Estimation of Survival Time Using a Failure Time Model

Variable	Coefficient estimate [95% CI]	$\chi^2(df)$	Survival multiplier
Age	0.006 [−0.003, 0.015]		
Female (vs. male)	−0.004 [−0.22, 0.21]		
Married	−0.299 [−0.498, −0.10]	8.65 (1) *	0.742
Education	−0.077 [−0.311, 0.157]		
Age of depression onset (0–23 as reference group)			
24–50	−0.053 [−0.451, 0.345]		
51 +	0.093 [−0.337, 0.522]		
Baseline HRSD-17 score	0.038 [0.011, 0.065]	7.75 (1) *	1.039
Other Axis I disorder	−0.143 [−0.341, 0.056]		
Pisa (vs. Pittsburgh)	−0.370 [−0.625, −0.115]	8.09(1)	0.691
Dimensional personality pathology	0.010 [0.003, 0.018]	7.88(1)	1.010

Note. HRSD-17 = Hamilton Rating Scale for Depression; CI = confidence interval; *df* = degrees of freedom.

*
 $p < .01$.